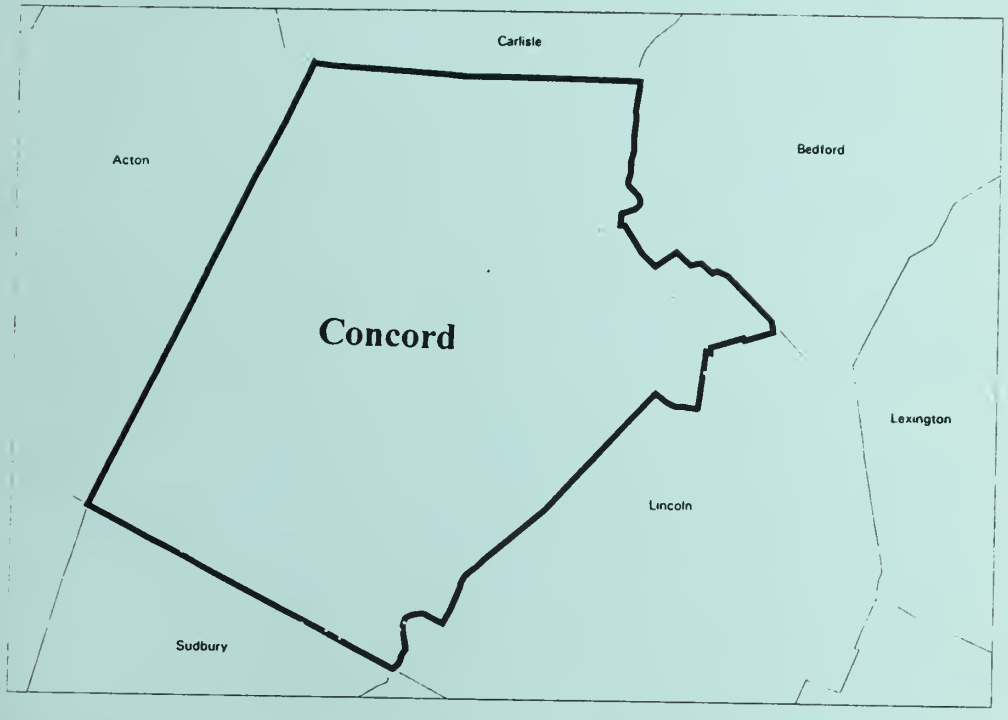
Health Consultation

Cancer Incidence 1982-1992
Concord, Massachusetts



Prepared by

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Bureau of Environmental Health Assessment
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I. INTRODUCTION

In response to requests by concerned citizens of Concord, Massachusetts, and the Concord Board of Health, the Community Assessment Unit of the Massachusetts Department of Public Health, Bureau of Environmental Health Assessment (MDPH/BEHA), conducted an investigation of cancer incidence in the town of Concord. This investigation is part of a response to a petition received by the Agency for Toxic Substances and Disease Registry (ATSDR) for the Nuclear Metals, Inc. (NMI) site in Concord.

The requests initiating this investigation originated primarily from environmental concerns specifically related to NMI, which is located at 2229 Main Street in Concord. These concerns focused primarily on the potential historical offsite migration and deposition of depleted uranium used at the NMI site. Such offsite migration would be a transport mechanism to offsite residents, who could then be exposed to uranium via inhalation or ingestion of the particulates.

This consultation addresses the following:

> What levels of radiation were reported from soil at offsite locations and what types of cancer are most likely associated with potential exposure to the radiological contaminant of most concern, uranium?
>
> Are there elevated rates of cancer in Concord or in smaller geographic areas of Concord?
>
> Do the reported levels of uranium in offsite soil suggest that increased incidence of cancer or noncancer effects may have resulted from opportunities for exposure to the uranium?
>
> If elevations in cancer rates exist, what further public health action or investigation may be warranted?

This report is organized by first providing background on the available radiological data for offsite soil sampling locations and a review of health effects associated with uranium. Next, the report reviews the incidence of nine cancer types (i.e., leukemia, multiple myeloma, and bone, brain, breast, colorectal, lung, prostate, and thyroid cancers) that may be associated with uranium exposures or that MDPH/BEHA was asked to evaluate in Concord and in each of its three census tracts (3611, 3612, and 3613) (Figure 1). The NMI facility is located in census tract 3612. In addition, the Concord Board of Health specifically asked MDPH/BEHA to review thyroid cancer incidence in each of the three towns adjoining Concord's census tract 3612 (Acton, Maynard, and Sudbury). The results of the cancer incidence review are then discussed, followed by conclusions and recommendations.

This investigation is descriptive in nature and can only provide a comparison of the incidence of the cancers in Concord with the incidence of these cancers in the state of Massachusetts. Descriptive assessments have certain inherent limitations. Only routinely collected data are analyzed and information about personal risk factors (e.g., family history, hormonal events, diet), which may influence cancer incidence is often limited and is not of an historical nature. It is beyond the scope of this investigation to determine any causal relationship or synergistic roles that the risk factors discussed in this report may have played in the development of cancer in Concord. The purpose of this investigation is to report our

findings and discuss them in the context of the available information to determine whether further investigation is warranted.

II. RADIOLOGICAL DATA FROM OFFSITE SOIL SAMPLING LOCATIONS

A. Radiological Data

Natural uranium in the environment contains uranium-238 (U-238), uranium-234 (U-234), and uranium-235 (U-235), which emit alpha particles and low levels of beta particles and gamma radiation and decay into other radionuclides, such as thorium, radium, and radon (ATSDR 1990). Natural uranium ores (e.g., pitchblende, phosphate rock) contain uranium, decay products, and other minerals (Weast 1977). Natural uranium extracted from the ore contains approximately 99.27 percent U-238 by weight, 0.72 percent U-235, and 0.0055 percent U-234. However, the decay products and other minerals have been removed. The radioactivity ratio of U-238, U-234, and U-235 is approximately 1.00:1.00:0.05, respectively (ATSDR 1990). Depleted uranium is the residual that remains after U-235 is mostly removed from natural uranium in the process of creating enriched uranium. During this enrichment process, U-235 increases to about 2-3 percent by weight for use in nuclear reactors, or to greater than 96 percent by weight for use in weapons and special reactors (ATSDR 1990). Depleted uranium has less than 0.7 percent U-235 by weight. If a uranium isotopic analysis is done on a sample, the uranium can usually be identified as natural, depleted, or enriched by the radioactivity ratio of U-238 to U-235. This activity ratio is approximately 22 for natural uranium and greater than 39 for depleted uranium (NRC and MDPH/RCP 1996).

NMI, which is licensed by the U.S. Nuclear Regulatory Commission (NRC) to possess radioactive materials, began operations at the Concord site in 1958 (Rocco 1983). The facility used depleted uranium to manufacture components for military, industrial, and medical applications (Rocco 1983). Thus, the predominant radioactive material used at the site was depleted uranium (O'Connell 1995, pers. comm.). A low inventory of highly enriched uranium (i.e., greater than 90 percent uranium-235) was kept on the site from 1968-1971 (Miller 1995, pers. comm.). The facility license for possessing enriched uranium was terminated in 1974 (O'Connell 1995, pers. comm.). Other radioactive materials that have been licensed or registered for use at the NMI according to NRC and Massachusetts Department of Labor and Industries records are natural uranium, thorium, cobalt-60, and cesium-137, the latter two of which are used for calibration and monitoring of equipment (O'Connell 1995, pers. comm.).

Several investigations of uranium and other radioactive materials have been conducted at NMI and offsite locations:

- In 1993 a local citizens' group, Citizens' Research and Environmental Watch (CREW) collected surface soil samples from six offsite locations. CREW reported results (CREW 1994a) for U-238 and radium-226. Samples were taken from about the top one to two inches of soil, and samples were analyzed by gamma spectral analysis. CREW also reported that one sample, taken in the Caterina Heights area, was analyzed by alpha spectral analysis to identify the ratio of uranium-238 to uranium-235 for the presence of depleted uranium.

- In 1994 the NRC and the MDPH Radiation Control Program (MDPH/RCP) collected surface and subsurface soil samples at 9 locations, including 7 offsite locations (see Figure 1) (MDPH 1995, NRC 1995). The surface soil sample volumes were collected from the 0-5 cm depth (or about 0-2 inches), and the subsurface soil samples were collected from the 5-25 cm depth. The offsite locations included samples from the Hillcrest Conservation land and the Caterina Heights area, areas that were also sampled by CREW. The two regulatory agencies split the samples and analyzed each sample at their own facilities. MDPH/RCP determined the concentrations of U-238, U-235, cesium-127, and potassium-40 in the soil samples. NRC determined the soil concentrations of U-235, U-238, thorium-232, bismuth-214, lead-214, radium-226, and cesium-137 in the samples. The results were reported in picocuries per gram (pCi/g). Analytical methods included gamma and alpha spectroscopy.

- NMI's consultant, Goldberg-Zoino and Associates (GZA), conducted soil sampling for total uranium in 1994 (Weidner 1995, pers. comm). GZA's samples were onsite samples, the locations of which were selected based primarily on MDEP modeling estimates of the areas of likely maximum impact from airborne emissions from the facility.

- In December 1995 the NRC and the MDPH/RCP collected 27 additional surface soil (0-2 inch depth) samples primarily within 0.5 mile of the NMI facility (see Figures 2 and 3). Background samples were also taken from locations at least five miles from the site. The samples were split between NRC, MDPH/RCP, NMI, and CREW. NRC and MDPH/RCP reported their results jointly (NRC and MDPH/RCP 1996).

For more details on the soil sampling programs, the respective groups who conducted the sampling should be contacted.

At the May 1995 public meeting, CREW reported that some of their offsite soil samples (all but one of which were taken within about 0.5 mile of the site) demonstrated elevated levels of uranium-238.

- Hillcrest Conservation land, near Kennedy's Pond—13.6 pCi/g
- 28 Maplewood Circle—2.4 pCi/g
- Maplewood Circle island—2.24 pCi/g
- NMI northeast fenceline (2 samples)—1.9 and 18.9 pCi/g
- Caterina Heights—16.4 pCi/g

CREW reported that depleted uranium was present at the Caterina Heights location based on their measurements of uranium-238 and uranium-235 and on a separate sample taken by the Harvard University Center for Isotope Geochemistry (CREW 1995).

Other results reported by CREW included concentrations of radium-226 ranging from 4.1 to 7.2 pCi/g (CREW 1994a). MDPH/RCP did not analyze for radium-226 because there are no records that the NMI facility had ever used radium (Hallisey 1995, pers. comm.). NRC did analyze for radium-226 (Miller 1995, pers. comm.) at all the offsite locations discussed above. NRC concluded that results for radium at offsite locations were consistent with natural background levels (Miller 1995, pers. comm.).

CREW (1994b) also reported that they determined, using a U.S. Environmental Protection Agency (EPA) model (CAP-88PC) that predicts airborne particulate migration from a site, that particulates emitted from the NMI facility could travel over one mile from the site (CREW 1994b). MDPH/BEHA is not aware of any other modeling conducted to predict the potential for airborne particulates to reach offsite locations. The MDEP calculations done to select soil sample locations for the MDEP Phase II investigation suggest that the maximum concentrations from stack emissions would occur at onsite locations and would likely result from aerodynamically induced downwashing (because the stack height to building height ratio is less than 1.5) (Mentos 1993, pers. comm.).

Results of the 1994 MDPH/RCP and NRC split samples were presented at a May 1995 public meeting held in Concord, which MDPH/BEHA staff attended. NRC and MDPH/RCP reported that their measurements from the split samples were in general agreement with each other (O'Connell 1995, pers. comm.). Both the NRC and the MDPH/RCP concluded that radioactivity measurements from offsite locations were consistent with background levels. One sample location reported by the NRC and the MDPH/RCP with results consistent with the presence of depleted uranium was discovered in the southeast corner of the NMI property (onsite), which suggests that the NMI facility has added to the natural background levels of uranium isotopes found in soil (O'Connell 1995, pers. comm.). Depleted uranium was detected at this location at about 11 pCi/g (NRC 1995) and about 20 pCi/g (MDPH/RCP).

GZA reported that most of the onsite samples taken during the Phase II investigation contained uranium above background concentrations (GZA 1995). The radioactivity measurements (for total uranium) ranged from 3.2 to 118 pCi/g for surface soil samples taken from 0-2 in at onsite locations (Weidner 1995, pers. comm.). MDEP considers site-specific background concentrations for total uranium to be approximately 1 pCi/g.

The NRC and the MDPH/RCP reported in their results of the December 1995 sampling that one type of analysis (gamma spectrometry) indicated that the majority of the 27 samples (including four of the five background samples) had positive results (i.e., detected values) for both uranium-238 and uranium-235. NRC and MDPH/RCP reported that the uranium in these samples indicated natural uranium when applying the U-238/U-235 ratio criteria. They also reported that all results from the gamma spectrometry method were indistinguishable from natural background (NRC and MDPH/RCP 1996). Another type of analysis (alpha spectrometry) on six sample locations and one background location identified depleted uranium in three samples located within 0.25 mile from the site. The total uranium in these samples ranged from 1.45 to 3.76 pCi/g. The background for total uranium ranged from 0.5 to 2 pCi/g, based on alpha spectrometry. Hence, the levels of depleted uranium were about 2 times natural background for uranium in soil (NRC and MDPH/RCP 1996).

B. Health Effects Associated with Uranium

The predominant radioactive material used at this site was depleted uranium metal. When uranium metal is exposed to air, it oxidizes and becomes uranium dioxide (UO_2) or triuranium octaoxide (U_3O_8), which are both insoluble in water (ATSDR 1990). Inhalation, oral, and dermal studies in animals have found that the toxicity of the uranium compound is dependent on the solubility of the compound in water. Soluble compounds are more readily absorbed into the body and into the bloodstream.

Whether airborne uranium particulates are inhaled and enter the respiratory tract is determined by numerous physical, chemical, and biological factors (ICRP 1993). The particle size and density, and its ability to absorb moisture from the atmosphere will determine the extent to which the material is inhalable. The physical and chemical properties also determine the penetration of the particle, the site of deposition, the retention times or clearance rates, and the rate of absorption into the blood and translocation to other tissue. For example, deposition of inhaled particulates with an aerodynamic diameter of 5 μm may be 82 percent for occupational male adult exposure but 34 percent would be deposited in the anterior nasal passage, 40 percent would be deposited in the posterior nasal passage and pharyns, 3 percent would be deposited in the bronchial tubes and bronchioles, and 5 percent would be deposited in the alveolar region. In contrast, deposition of inhaled particles with an aerodynamic diameter of 1 μm may be 47 percent for environmental male adult exposure (lighter activity), but 14 percent would be deposited in the anterior nasal passage, 18 percent would be deposited in tht eposterior nasal passage and pharynx, 3 percent would be deposited in the bronchial tubes and bronchioles, and 12 percent would be deposited in the alveolar region (ICRP 1993). Insoluble uranium that reaches the alveoli of the lungs may be removed after several months by ciliary action, swallowed, and excreted through the gastrointestinal tract (BEIR 1988). Insoluble uranium greater than 5 μm cannot penetrate as far as the alveoli and are usually removed within hours or days, swallowed, and excreted through the gastrointestinal tract. Thus, the less soluble uranium compounds are largely cleared from the lungs and eliminated through the gastrointestinal tract (BEIR 1988). Some of the uranium remaining in the lungs will enter the blood, pass through the kidneys, and be eliminated in the urine within a few days. Some may stay in the lungs for years. Since UO_2 and U_3O_8 are insoluble, they are more likely to be coughed or breathed out or retained in the lungs rather than absorbed into the blood. Thus, a very small fraction would be absorbed into the blood. Animal studies have not indicated short-term exposure effects (14 days or less) for insoluble uranium compounds (ATSDR 1990). Adverse health effects on the respiratory system have been observed in humans and animals following chronic exposure to insoluble uranium dust at high levels (5 mg uranium/m^3 of air), which is 25 times higher than levels allowed in an industrial environment and approximately 250 times higher than the average concentration of all particulates measured in outdoor air in Middlesex County, Massachusetts in 1995 (U.S. EPA 1995).

Most ingested uranium is excreted in the feces. A small fraction (0.2 percent for insoluble compounds) is absorbed into the blood (ICRP 1990). Of the amount absorbed into the blood, 10 to 20 percent is retained in the kidneys, approximately 10 to 30 percent is retained by the bone, and the remainder is excreted in the urine (ATSDR 1990, BEIR 1988). The amount retained in the kidneys has undergone chemical changes in the blood, reacts chemically at the cellular level in the kidneys, and may cause cellular damage. It usually clears the kidney within a few days, and the tubule cells begin to regenerate; however, this does not mean there is no subtle functional damage (ATSDR 1990). One μg uranium/g of renal tissue may be a threshold for nephrotoxicity, but this threshold level is a matter of controversy and could be lower than 1 μg/g renal tissue (BEIR 1988). A 1 μg/g threshold value would correspond to a total uranium content of 310 μg (or approximately 210 pCi) for two kidneys in an adult man (Wrenn et al. 1985). This would correspond to an adult man ingesting 25 to 30 kilograms (or about 55-66 pounds) of soil containing about 20 pCi uranium/g of soil, which is slightly higher than the maximum concentration reported in any offsite location in NMI-related investigations (18.9 pCi/g).

For radiobiological effects, human and experimental animal studies have not conclusively linked natural or depleted uranium exposure to cancer (Wrenn et al. 1985), but animal studies have shown that highly

enriched soluble uranium (absorption fraction of 5 percent) can produce bone sarcomas in rats (BEIR 1988). In addition, other alpha-emitting radionuclides (e.g., radium-224, radium-226, radium-228, radon 222, polonium-210, polonium-214, and polonium-218) have been linked to bone and lung cancers (ATSDR 1990, BEIR 1988). Alpha particles are a type of ionizing radiation, which is defined as any radiation capable of displacing electrons from atoms or molecules, thereby producing ions (BEIR 1990). The other principal types of ionizing radiation include beta particles, x-rays, and gamma radiation. Alpha particles are unable to penetrate the skin, but can travel short distances in the body and cause damage if they are inhaled or ingested. Beta particles are smaller than alpha particles. External beta particles can be hazardous if the radiation is within a few centimeters of exposed skin. Internally, beta particles inflict less damage than alpha particles. Gamma radiation can penetrate the skin and the energy is deposited in or passed through the body. Natural uranium emits primarily alpha particles and a low level of beta particles and gamma radiation (ATSDR 1990). Depleted uranium is also primarily an alpha emitter.

Ionizing radiation is clearly linked to many types of cancer (BEIR 1990, ATSDR 1990). However, most cancers associated with exposure to ionizing radiation are from radiation (e.g., x-rays) administered during treatment for a variety of medical conditions or from external radiation (e.g., gamma radiation, beta particles) originating from explosions of atomic bombs or fallout from nuclear weapons testing. Other evidence includes varying strengths of association between occupational exposures to ionizing radiation and certain types of cancers (e.g., bone cancer in radium dial workers; leukemia and multiple myeloma in nuclear facility workers) (Schottenfeld and Fraumeni 1996).

Studies of uranium miners exposed to alpha-emitting radon daughters (e.g., polonium-210, polonium-214, and polonium-218), showed increased risks of lung cancers (Amdur et al. 1991, ATSDR 1990, BEIR 1990). Studies of radium dial painters exposed to soluble radium-226 or radium-228 (absorption fraction for radium of 20 percent versus 0.2 percent for oxidized uranium) have shown an association with bone sarcoma (Amdur et al. 1991, BEIR 1988, ICRP 1990). Based on these studies, particularly of radium exposures, it is reasonable to assume that highly enriched soluble uranium, which is more radioactive than natural or depleted uranium, has the potential to increase the risk of bone sarcomas in humans (BEIR 1988). Uranium and radium are deposited in mineral bone (the location for bone sarcomas) and not in the red marrow (the location of cells which give rise to leukemias). The risk of radiation-induced leukemia in humans has been low relative to the risk from bone sarcoma for alpha emitters in mineral bone (Mays et al. 1985).

Multiple myeloma was observed in six of the 1,285 radium dial painters. Of these six cases, four women worked as dial painters for at least 50 weeks, one worked only two weeks, and the duration of employment for the remaining worker is unknown. If the four excess cases of myeloma were caused by skeletally deposited radium, induced myeloma would be only 6 percent as frequent as the number of bone sarcomas. In a German study there was only one case of multiple myeloma among 2,324 patients injected with radium-224. Fifty-five of these patients developed bone sarcomas. These studies indicate that bone sarcomas and carcinomas of the paranasal sinuses and mastoid air cells are the vast majority of radium-induced cancers. (The latter category is believed to be related to the decay of radon-222 gas and is not observed with radium-224 exposures.)

The risk for uranium-induced bone sarcomas from daily ingestion of 5 pCi U/day over a 70-year lifetime would be about 1.5 cases in 1 million persons exposed. In contrast, based on estimates of prevalence in

the general population of bone sarcomas, about 750 bone sarcomas are expected among these 1 million persons during their lifetimes (Mays et al. 1985).

Although human or animal studies do not conclusively link natural or depleted uranium exposure to cancer, this review indicates that bone sarcomas and lung cancer are the cancer types that appear to be most likely associated with potential exposures to natural or depleted uranium.

III. CANCER INCIDENCE ANALYSIS

A. Methods for Analyzing Cancer Incidence Data

The term cancer is used to describe a variety of diseases associated with abnormal cell and tissue growth. These different diseases are classified by primary site (location where the disease originated) and histology, or cell type. Epidemiologic studies have revealed that different histologic types of cancer are individual diseases with separate causes, risks, characteristics, and patterns of survival.

Cancer incidence data for the years 1982-1992 were obtained for the town of Concord from the Massachusetts Cancer Registry (MCR) of the MDPH Bureau of Health Statistics, Research and Evaluation. As previously noted, despite the fact that the cancers of primary concern (relative to uranium) include lung and bone cancer, residents also asked the MDPH/BEHA to analyze seven other cancer types in Concord. In addition, because of a request by the Concord Board of Health, cancer incidence for thyroid cancer for three towns that are adjacent to Concord's census tract 3612 (i.e., Acton, Maynard, Sudbury) were also obtained. The MCR has been monitoring cancer incidence in the Commonwealth since 1982. The eleven-year period from 1982 to 1992 is the most recent for which complete data exist. To determine whether an elevated rate of a specific type of cancer exists in Concord, cancer incidence data were adjusted by age and sex and analyzed to compare the actual (or observed) number of cases to the number that would have been expected based on the statewide cancer incidence experience.

In order to calculate incidence rates, it is necessary to obtain accurate population information. The population figures used in this analysis were interpolated based on 1980 and 1990 census data for Massachusetts (U.S. DOC 1980, 1990). To estimate the population between census years, an assumption was made that the change in population occurs at a constant rate throughout the ten-year interval between each census. From these calculations, 1987 population estimates for the city of Concord and each of the three Concord census tracts were obtained. Population estimates for the year 1987 were used to calculate SIRs for each of the three time periods evaluated.

Standardized incidence ratios (SIRs) were calculated for the nine cancer types for the period 1982-1992 for Concord as a whole and for each of three census tracts in Concord (NMI is located in census tract 3612). In order to evaluate temporal trends in cancer incidence, SIRs were also calculated for the two time periods 1982-1986 and 1987-1992. SIRs were not calculated when fewer than five cases were observed because the rates would be very unstable due to the small numbers. Available risk factor information, as reported to the MCR (e.g., smoking status, occupation) was also reviewed.

An SIR is an estimate of the occurrence of disease in a population in relation to what might be expected if the population had the same cancer experience as some larger population designated as "normal" or average. Usually the state as a whole is selected to be the "normal" population.

Specifically, an SIR is the ratio of the observed number of cancer cases to the expected number of cases. An SIR of 100 indicates that the number of cancer cases in a given population is equal to the number of cancer cases expected in a normal population. An SIR greater than 100 indicates that more cancer cases occurred than expected; an SIR less than 100 means that fewer cases occurred than expected. Accordingly, an SIR of 150 is interpreted as 50 percent more cases than the expected; an SIR of 90 indicates 10 percent fewer cases than expected.

Caution should be exercised, however, when interpreting an SIR. The interpretation of an SIR depends on both the size and the stability of the SIR. Two SIRs can have the same size but not the same stability. An SIR of 150 based on two expected cases and three observed cases indicates a 50 percent excess in cancer, but this high percentage of excess is based on one case, which may have occurred due to chance alone. Conversely, an SIR of 150 based on 200 expected cases and 300 observed cases shows the same 50 percent excess in cancer, but because the SIR is based on a greater number of cases, the estimate is more stable. It is very unlikely that 100 excess cases would occur due to chance alone.

To determine if the observed number of cases is statistically significantly different from the expected number or if the difference may only be due to chance, a 95-percent confidence interval is calculated (Rothman and Boice 1982). A 95-percent confidence interval is the range of estimated SIR values that has a 95 percent probability of including the true SIR for the population. If the range does not include the value 100, then this means that the study population is significantly different from the normal population and there is a 5 percent chance that the observed difference is merely the result of chance variation in the number of observed cases. If the range excludes 100 and the observed SIR is greater than 100, then the number of cancer cases is considered significantly higher than expected. Similarly, if the range excludes 100 and the observed SIR is less than 100, then the number of cancer cases is significantly lower than expected. If the range includes 100, then this means the true SIR may be 100, and we cannot conclude with sufficient confidence that the observed SIR reflects a real cancer excess (or deficit).

The width of the confidence interval reflects the stability of the SIR value. For example, a narrow confidence interval (such as 103-115) allows a fair level of certainty that the observed SIR is close to the true SIR value for the population. A wide interval (e.g., 82-450), on the other hand, leaves considerable room for doubt about what the true SIR is. That is, the true SIR could be much higher or much lower than the observed SIR.

The geographic distribution of cancer cases was determined using available information regarding the place of residence at diagnosis. This information was mapped for each individual using the computer mapping software MapInfo (MapInfo 1994).

The available smoking status and occupational information were analyzed when pertinent to the cancer type. These data were also evaluated in relation to the geographic distribution of cancer cases. While these are the most complete and accurate data readily available, there were many cases where the smoking status and the occupation of the patient were not reported, and hence, these data are incomplete.

The observed number of cases reported in the following section for Concord are in some instances slightly different from the observed number of cases shown in the MCR report, *Cancer Incidence in Massachusetts 1982-1992 City and Town Supplement* (MDPH 1995). The data contained in MDPH (1995) reflect information entered in the MCR computer files prior to the date this file was closed and made available for research. The data in this research file are constantly being quality controlled so that corrections may be made in subsequent reports. Occasionally, the research file may contain duplicate cases.

The data discussed in this report have been controlled for duplicate cases. The analyses account for duplicate cases by removing the duplicate cases while including multiple primary cancer cases. A multiple primary cancer case is defined by the MCR as a new cancer of the same histology as an earlier cancer, if diagnosed in the same site more than two months after the initial diagnosis (MCR 1996). Duplicate cases are additional reports of the same primary site cancer case. The decision that a case was a duplicate and should be excluded from the analyses was made by the MCR after consulting with the reporting facilities and obtaining additional information regarding the histology or pathology of the case.

In addition, the MCR compiles and reports tumors originating in the central nervous system as one group. This group of tumors includes malignant brain tumors, spinal cord tumors, nerve sheath tumors, and benign tumors of the brain and spinal cord. Because of differences in risk factors associated with primary brain cancer and other central nervous system sites (the majority of which develop due to congenital disorders), for the purpose of this report, only primary brain tumors have been analyzed. Primary brain tumors are those tumors that arise from the brain and its coverings. Metastatic brain tumors, which were not included in this analysis, are the result of other types of cancers spreading to the brain (Black 1991a).

B. Cancer Incidence in Concord

Tables 1 through 12 summarize the cancer incidence data townwide and for each census tract for three different time periods: 1982-1986, 1987-1992, and for the entire 1982-1992 period. Figure 1 depicts the three census tracts and the NMI site, which is in census tract 3612. Table 13 summarizes the thyroid cancer incidence data for Acton, Maynard, and Sudbury. The following sections present results for Concord as a whole and then for each of the three census tracts. The census-tract-specific analyses help in understanding whether the incidence of cancers observed for the town may be at least partially explained by some particular geographic distribution of the cases within the town.

Cancer Incidence for Concord as a Whole (Tables 1-3)

For males and for males and females combined, lung cancer for all three time periods occurred statistically significantly less often than expected for Concord as a whole. Among females, lung cancer occurred less often than expected for all three time periods.

One case of bone cancer occurred in Concord during the 1982-1986 period (versus slightly less than one expected), and no bone cancers were reported for Concord during the 1987-1992 period.

Thyroid cancer was significantly elevated in the total population for the 1982-1992 period (16 obs vs 6.9 exp). It was also significantly elevated females (10 obs vs 4.6 exp) for this 1982-1992 period, and

elevated but not statistically significant for males (6 obs vs 2.3 exp). When analyzed by the two smaller time periods, thyroid cancer was significantly elevated among males and females combined (10 obs vs 4.0 exp) in the 1987-1992 period. In 1982-1986, thyroid cancer was elevated among males and females combined (6 observed vs 2.9 expected), but this elevation did not achieve statistical significance.

Multiple myeloma among females occurred at approximately the rate expected or less often than expected for all three time periods. Among males, multiple myeloma occurred about as expected in 1982-1986, but was significantly elevated from 1987-1992 (9 obs vs 2.2 exp), which explained the 1982-1992 significant elevation among males for this cancer (10 obs vs 3.9 exp).

Brain cancer among males occurred about as expected for all three time periods in Concord. Among females, an excess of approximately 2 cases occurred during 1982-1986 (4 obs vs 2.3 exp), but brain cancer was significantly elevated in females for the 1987-1992 period (9 obs vs 3.2 exp), which primarily explained the significant elevation among Concord females for the entire 1982-1992 period (13 obs vs 5.4 exp).

Female breast cancer was significantly elevated in Concord from 1982-1986 (82 obs vs 56.2 exp), but occurred slightly less than expected in the 1987-1992 period (75 obs vs 78.5 exp). The 1982-1986 significant elevation accounted for why female breast cancer for the entire 1982-1992 period nearly achieved statistical significance.

In all three time periods, leukemia incidence was elevated among Concord residents, but no elevation was statistically significant. The excesses for all time periods ranged from about 1 to 5 cases among males and 1 to 3 cases among females.

Prostate cancer among Concord males occurred less often than expected during the 1982-1986 period, but more often than expected during the 1987-1992 period (73 obs vs 56.7 exp). This latter elevation was statistically significant. For the 1982-1992 period, prostate cancer was elevated (98 obs vs 89.7 exp), but this elevation was not statistically significant.

Colorectal cancer in Concord as a whole occurred about as expected for all three time periods and for both sexes.

Census Tract 3611 (Tables 4-6)

Lung cancer incidence occurred less often than expected for all three time periods and for both sexes. These rates achieved statistical significance for males and for males and females combined for the 1987-1992 and 1982-1992 time periods. No bone cancer cases were reported for this census tract.

Thyroid cancer in males and females combined was significantly elevated for the 1987-1992 time period (5 obs vs 1.5 exp), primarily explained by an elevation in females (4 obs vs 1.0 exp). During 1982-1992 thyroid cancer was significantly elevated among females (6 obs vs 1.7 exp) and occurred about as expected among males (1 obs vs 0.9 exp).

Multiple myeloma in males of census tract 3611 occurred about as expected in 1982-1986, but was elevated in 1987-1992 (4 obs vs 0.9 exp), which primarily accounted for the significant elevation among males for the 1982-1992 period (5 obs vs 1.5 exp). No multiple myeloma was reported for females in this census tract.

A total of 5 brain cancer cases, which was about as expected (4.7 cases), occurred in this census tract during 1982-1992. Four of these cases occurred among females, which was two more cases than expected.

Female breast cancer was elevated in all three time periods (e.g., during 1982-1992, 66 obs vs 54.4 exp), but no elevation was statistically significant. One male breast cancer case occurred in this census tract during 1982-1992.

For leukemia, in general, about 1 to 2 more cases occurred than expected for each sex for both smaller time periods and the overall period (e.g., for males during 1982-1992, 5 obs vs 3.5 exp).

Prostate cancer occurred about as expected during the 1982-1986 period. It was elevated during 1987-1992 (31 obs vs 22.7 exp), but this elevation was not statistically significant.

Colorectal cancer occurred less often than expected for all time periods and for each sex.

Census Tract 3612 (Tables 7-9)

Lung cancer occurred about as expected or less often than expected in this census tract for all time periods and for both sexes. One case of bone cancer (female) occurred in the 1982-1986 period, but no cases occurred during 1987-1992.

No thyroid cancer cases occurred in this census tract during 1982-1986. Four cases (versus 1.1 exp) occurred during 1987-1992, with 2 cases in males and 2 cases in females.

Multiple myeloma occurred about as expected during 1982-1992 (2 obs vs 1.7 exp; one male and one female case).

Brain cancer in females was significantly elevated during 1982-1992 (5 obs vs 1.3 exp), which was attributed primarily to the elevation in females during 1987-1992 (4 obs vs 0.8 exp). Brain cancer occurred about as expected for males during all time periods.

While female breast cancer was significantly elevated in this census tract during the 1982-1986 time period (24 obs vs 12.9 exp), it occurred less often than expected during 1987-1992 (12 obs vs 18.3 exp). During 1982-1992 female breast cancer was elevated (36 obs vs 31.2 exp) but was not statistically significant. One case of breast cancer in males occurred during this time period.

Leukemia occurred about as expected among males in this census tract. Among females, leukemia occurred more often than expected during 1982-1992 (4 obs vs 1.5 exp), which was attributed to an elevation during the latter 1987-1992 time period (4 obs vs 0.8 exp).

Prostate cancer occurred less often than expected during 1982-1986 (4 obs vs 7.1 exp), more often than expected (but not statistically significantly elevated) during 1987-1992 (17 obs vs 13.1 exp), and about as expected during the entire 1982-1992 period (21 obs vs 20.6 exp).

Colorectal cancer among both sexes combined occurred at about the rate expected during 1982-1986 (13 obs vs 13.2 exp). Colorectal cancer occurred more often than expected during 1987-1992 (19 obs vs 14.9 exp; not statistically significant), and slightly more often than expected during the entire 1982-1992 period (32 obs vs 28.1 exp).

Census Tract 3613 (Tables 10-12)

Lung cancer occurred less often than expected for all three time periods and for both sexes. During 1987-1992 as well as for the entire 1982-1992 period, the rates of lung cancer were statistically significantly lower than expected for males. No bone cancer cases occurred in this census tract.

Thyroid cancer was elevated during the 1982-1992 period (5 obs vs 2.5 expected), but this elevation was due to about 2 excess cases and was not considered statistically significant. Four of these cases (2 of each sex) occurred during 1982-1986 (versus 1.0 expected for males and females combined).

About 1 excess case of multiple myeloma (4 obs vs 2.8 exp) occurred during the entire 1982-1992 period in this census tract. Three of the 4 cases occurred in males during 1987-1992 (versus 0.8 expected).

Brain cancer occurred about as expected during 1982-1986 and was elevated during 1987-1992 (6 obs vs 2.6 exp; evenly distributed between sexes). This latter elevation was not statistically significant.

Female breast cancer was elevated during 1982-1986 (29 obs vs 20.5 exp), but occurred slightly less than expected during 1987-1992 (26 obs vs 28.8 exp). No cases of male breast cancer occurred in this census tract during 1982-1992.

Leukemia in males was significantly elevated in 1982-1986 (5 obs vs 1.5 exp) but occurred about as expected during 1987-1992. This resulted in an elevation that was not statistically significant during the 1982-1992 period among males (7 obs vs 3.3 exp). No cases of leukemia occurred among females in this census tract.

Prostate cancer occurred less often than expected during 1982-1986, more often than expected during 1987-1992, and about as expected during the entire 1982-1992 period.

Colorectal cancer was elevated during 1982-1986 (27 obs vs 22.5 expected; not statistically significant) and occurred about as expected during 1987-1992. Overall, during 1982-1992 colorectal cancer showed a slight elevation in this census tract (53 obs vs 47.8 exp).

Geographic Distribution

Place of residence of individuals at the time of diagnosis was evaluated. (Two lung cancer cases, one multiple myeloma case, and one prostate cancer case could not be located as to their census tract of

residence due to incomplete address information. However, these cases were included in the analyses for the town as a whole.) In addition to determining census-tract-specific incidence ratios for all cancer types, MDPH/BEHA staff qualitatively evaluated whether any specific cancer type appeared to be concentrated in some area(s) within each census tract. In the case of breast cancer, the MDPH/BEHA breast oncologist consultant also examined the geographic distribution of cases.

With one exception, no concentration of any specific type of cancer occurred within any census tract that is not likely attributed to the presence of a multiunit complex, a nursing home, or more densely populated areas within the census tracts. The exception was male multiple myeloma incidence in census tract 3611, where some cases appeared to be concentrated in one geographic area. This area is approximately 3-3.5 miles from the NMI site.

Two cancer types were significantly elevated in census tract 3612. Female breast cancer for 1982-1986 and female brain cancer for 1982-1992 (attributed to an elevation during 1987-1992) were significantly elevated in this census tract. However, neither of these cancers appeared concentrated in specific areas within 3612.

Occupational Data

As stated earlier, some cases reported to the MCR do not contain meaningful occupational information (e.g., occupation may be listed as "retired"). In other cases, no information is provided. Thus, occupational information on the Concord cases is generally of limited use. MDPH/BEHA staff reviewed all available occupational information for those cases with cancers that were significantly elevated for any time period, for Concord or any census tract, or for either sex. The information reviewed did not allow for a determination to be made on the role of occupation in explaining elevated cancer incidences in Concord.

One concern that has been raised by CREW members is whether Concord residents who contracted leukemia may have previously worked at NMI. Due to patient confidentiality laws, we cannot actually say how many people worked at NMI due to the potential for case identification. However, the available occupational information in the MCR database for the 22 leukemia cases that occurred in Concord during 1982-1992 showed that 8 individuals did not have any occupational information and 3 individuals provided an occupational title (but no company name) that could indicate employment in many different types of companies. Three individuals were children or students, and 8 individuals named a professional affilitation or field that did not suggest a professional affiliation with NMI. Thus, overall, data are insufficient to evaluate possible professional affilitations with a specific company, such as NMI, for half of the leukemia cases.

Thyroid Cancer in Acton, Maynard, and Sudbury

Table 13 summarizes the thyroid cancer incidence for the three towns adjoining census tract 3612. The number of cases was too small to evaluate statistical significance in most cases. In Acton 9 cases occurred during 1982-1992, while approximately 7 were expected. Most of these Acton cases occurred during 1987-1992, and the elevation in males during this time period was statistically significant (5 obs vs 1.1 exp), while the incidence in females was about as expected. In Maynard, about 2 fewer cases

occurred than expected during the 1982-1992 period (2 obs vs 4.1 exp), while in Sudbury about 1 more case occurred than expected during the same time period (6 obs vs 5.4 exp).

The nearest residence of any thyroid cancer case in these adjoining towns was about one mile from the NMI site. Thyroid cancer did not exhibit unusual concentrations in any specific areas of any of the three towns.

IV. DISCUSSION

While a number of cancer types were statistically significantly elevated in Concord as a whole and in certain census tracts (Table 14 and Figure 4), these cancers were not the types that appear to be most likely associated with the primary radiological contaminant of concern, that is, depleted uranium. Of all cancer types, lung cancer and bone sarcoma are the cancer types that are most likely to be associated with exposure to natural or depleted uranium. Lung cancer occurred about as or less often than expected for all time periods, all census tracts, and both sexes; some rates were statistically significantly lower than expected. One bone cancer case (female) was reported for Concord. This individual resided in census tract 3612. The residence reported for this individual was over one mile from the NMI site.

As to the other seven cancer types investigated here (thyroid, multiple myeloma, brain, breast, prostate, colorectal), thyroid cancer was most consistently elevated in terms of elevations in both time periods and among both males and females. The only other cancer that presented an unusual picture was mutliple myeloma for which some cases appeared to be concentrated in one area of Concord.

The types of cancers significantly elevated in Concord and evidence regarding other types of ionizing radiation or risk factors are discussed below.

Thyroid Cancer

The incidence of thyroid cancer in general is very low (ACS 1996). It is, however, one of the commonest neoplasms in adolescents and young adults, and its incidence is about three times greater in women than in men (Higginson et al. 1992). It is primarily associated with external x-ray treatments of benign medical conditions in childhood (U.S. EPA 1989, Amdur et al. 1991, BEIR 1990, ACS 1996), or, in the case of the atomic bomb survivors, whole-body external radiation such as gamma radiation (e.g., Ron et al. 1995, Lundell et al. 1994, BEIR 1988). An analysis of data from the Marshall Islands on radioactive fallout exposures also show an association between radiation exposures (i.e., external gamma radiation, inhaled or ingested radioiodides, which were mostly [73-96 percent] beta particle emitters) and elevated thyroid cancer incidence (BEIR 1990). Therapeutic use of radioactive iodine (primarily for treatment of thyrotoxicosis) does not appear to be associated with an increase in thyroid cancer risk (BEIR 1990; Higginson et al. 1992; ACS 1996). Additional risk factors for thyroid cancer are non-malignant thyroid disorders and a positive family history of these disorders (Higginson et al. 1992).

Sixteen thyroid cancer cases occurred in Concord residents from 1982-1992. More cases (10) occurred in females than males (6). Ten of the cases occurred in individuals less than 40 years old.

Female Breast Cancer

While age-adjusted mortality rates for breast cancer have remained constant since 1930, breast cancer incidence rates have been rising over the past several decades (Kelsey and Gammon 1990). The incidence of female breast cancer in Massachusetts has been increasing since 1982 with some leveling off occurring in the late 1980s (MDPH 1993). A portion of the increase in incidence is likely attributed to early detection brought on by mammography use and self exam. However, improved detection is not likely to be the only factor associated with the increase in incidence rates. The identified risk factors account for less than half of the reported breast cancer cases in the United States. Thus there is speculation concerning other potential risk factors (Flack 1992).

According to the American Cancer Society (ACS 1996), breast cancer is the second major cause of cancer deaths in women. Breast cancer deaths are surpassed only by lung cancer deaths in females. More cases of breast cancer in women are diagnosed each year than any other cancer (Kelsey and Gammon 1991). There are several known risk factors for breast cancer and many that are under current investigation.

The risk of breast cancer in females increases over the age of 40, with the majority of cases occurring greater than age 50. The major known risk factors for breast cancer are attributed to genetics and hormones. A family history of breast cancer, early onset of menstruation, late age at menopause, never having had children, late age at first full-term pregnancy, higher education, and higher socioeconomic status have been identified as risk factors for breast cancer in females. There appears to be some variation in risk factors depending upon pre/post-menopausal status. Some studies indicate that obesity appears to be a risk factor in postmenopausal women while thin premenopausal women appear to be at a higher risk. Evidence from animal and human epidemiologic data suggest that dietary fat may play a key role in the development of breast cancer (Boyd et al. 1992). Previous studies have suggested that women of Jewish descent might have a higher rate of breast cancer than non-Jewish women (Kelsey and Gammon 1991).

Higher socioeconomic status and higher education are believed to be correlated with risk factors for an increased risk of breast cancer. Possible explanations for this increased risk include overnutrition and delayed childbearing in higher socioeconomic populations. The incidence may appear elevated in higher socioeconomic populations due to an increased detection rate. These populations may have been access to medical care and thus may be more likely to seek screening for breast cancer.

The risk of breast cancer is increased in women with a history of breast cancer, endometrial cancer, or ovarian cancer (Kelsey and Gammon 1990). Women with a history of primary breast cancer are believed to have a three to four-fold increase in the risk for primary cancer in the contralaterial breast (Kelsey and Gammon 1991).

The relationship between breast cancer and the use of hormones both for contraception and as menopausal replacement therapy, has been intensely studied. There is some evidence of an increased breast cancer risk in women who have used replacement estrogens. Long-term replacement estrogen users seem to be a particular risk, although the available data are not consistent. Recent metanalyses (pooled studies) have suggested that the increased risk of breast cancer from estrogen replacement for a period of 5 to 10 years is nonexistent or extremely small. No conclusive evidence is available to link oral contraceptive use with

increased risk of breast cancer (Page and Asire 1985).

Women with family histories of breast cancer in close relatives have an increased risk of breast cancer. Recent scientific research has identified genes related to breast cancer. These genes are BRCA1, BRCA2, and p53, which can be inherited in forms capable of conferring increased breast cancer susceptibility. Typically, individuals are born with two copies of every gene. When a mutated form of one of these genes is inherited, there may be a marked increase in breast cancer susceptibility. These genes are believed to act as tumor suppressors (i.e., their function is to suppress the formation of tumors). If an individual inherits a mutation which alters its tumor suppressor capacity, only one copy of the tumor-suppressor gene is acting to suppress tumor formation. If this one active copy is then inactivated by a noninherited event, the chance of developing cancer increases. Thus, women who inherit mutant versions of this gene are at higher risk of developing breast cancer than women who do not carry mutated versions of the gene (Harris et al. 1996).

Ataxia-telangiectasia is an autosomal recessive genetic disorder (i.e., a disorder which requires two defective genes). This disorder is characterized by cerebellar ataxia, oculocutaneous telangiectasias, radiation hypersensitivity, and an increased incidence of malignant disease. Preliminary observation indicates that women presumed to be heterozygous (inherit one defective copy and one nondefective copy of the gene) for ataxia-telangiectasia have excess breast cancer (Harris et al. 1996).

While definitive information about the role of environmental factors in breast cancer is not available, studies are currently underway to evaluate potential links between breast cancer and environmental factors such as pesticides and electromagnetic fields (Kelsey and Gammon 1990; Flack 1992). The primary environmental risk factor that has been identified in relation to breast cancer is exposure to ionizing radiation, as evidenced by followup studies (e.g. Tokunaga et al. 1987) of atomic bomb survivors exposed to external whole body radiation, such as gamma radiation, and from studies of patients treated with x-rays for various medical conditions (e.g., Davis et al. 1989). One recent study conducted by researchers at the Mount Sinai School of Medicine in New York has suggested a possible association between breast cancer and exposure to certain pesticides (Wolff et al. 1993, Krieger et al. 1994). This study, however, was of a preliminary nature and not conclusive in its results.

The MDPH/BEHA is presently conducting research initiatives exploring a potential relationship between breast cancer and environmental agents, including a major study, conducted by a contractor to the MDPH, on the environment and breast cancer on Cape Cod and a pilot study conducted solely by MDPH staff in Berkshire County, Massachusetts, investigating changes in serum PCB and pesticide concentrations over time in breast cancer patients. The Berkshire study aims to provide important information on data gaps identified from the Mount Sinai study by recruiting females within weeks of their diagnosis. The primary data gaps identified in the Mount Sinai study included the recruitment of female breast cancer patients after diagnosis and treatment had been initiated. This raised the question of whether the association observed between diagnosis of breast cancer and body burdens of PCBs/DDE were an artifact related to physiologic change after diagnosis and treatment or whether the observed association was real.

Multiple Myeloma

Multiple myeloma has become increasingly common in recent years (Higginson et al. 1992). It generally occurs late in life, with a median age of diagnosis in the U.S. of 68 years in males and 69 years in females (Higginson et al. 1992). Radiation exposure is the only known risk factor so far identified for multiple myeloma (Higginson et al. 1992), as evidenced from an increase among radiologists and atomic bomb survivors (BEIR 1990), both of which reflect external radiation exposures. Nuclear power plant workers may also be at increased risk (NCI 1996). In addition, case-control studies in various populations have consistently reported an increased risk associated with farming or agricultural work but the reason remains unknown (Higginson et al. 1992). Among chemical agents, there is some evidence of an association between duration of exposure to benzene and other solvents and multiple myeloma (Higginson et al. 1992). Experimental observations and a few case reports support the hypothesis that the risk of multiple myeloma may result increase after prolonged stimulation of the immune system by repeated infection, allergic conditions, or autoimmune disease (ACS 1996, NCI 1996). Also, there is some evidence for genetic predisposition for multiple myeloma (NCI 1996).

Brain Cancer

Little is known regarding the etiology or causes of brain cancer. About 52 percent of primary brain tumors in adults are glioblastomas, 18 percent meningiomas, and 10 percent astrocytomas (Higginson et al. 1992). Among children, brain cancer is the second most common cancer.

Risk factor information for brain cancers varies depending on the histology of the cancer. Sixteen percent of those with primary brain tumors have a family history of cancer in the family (Black 1991b). Astrocytoma has been associated with the development of multiple sclerosis in adults and exposure to lead in children. Glioblastoma has been associated with several viruses and occupational exposure to vinyl chloride. Some studies have noted a genetic pattern or familial tendency to develop gliomas (glioblastomas and astrocytomas are both gliomas); most commonly documented is the occurrence of glioblastoma among families (Farwell and Flannery 1984; Mauron et al. 1984; Salcman and Solomon 1984). Both astrocytoma and glioblastoma have been associated with cranial irradiation. Other factors that may be related to brain cancer include high-dose x-rays (e.g., prenatal x-ray exposures may lead to brain tumors (NRC 1990; Higginson et al. 1992), consumption of sodium nitrate (meat preservative), head trauma, exposure to some occupations (e.g., refinery of crude oil and production of petroleum-based chemicals, nuclear fuels and weapons industry, farmers, manufacture of synthetic rubber and polyvinyl chloride) (MCI 1996), and the use of barbituates by pregnant women and by children (Page and Asire 1985).

Meningioma is a slow growing, usually benign tumor which has a high survival rate. Suspected risk factors in the development of meningiomas include previous head trauma (including injury and surgery), hormonal factors, certain genetic disorders, and exposure to some chemicals (Black 1991b).

The human data for the relationship between brain cancer and ionizing radiation are derived from studies of populations exposed prenatally to diagnostic x-rays (see above) and populations exposed postnatally to therapeutic x-rays or atomic bomb radiation (BEIR 1990). These radiation-related reports include increased risk of brain tumors in children who received scalp radiation for tinea capitis in Israel and other x-ray treatments of head and neck during childhood (BEIR 1990), increased risks reported for workers in a nuclear fuel fabrication plant (Higginson et al. 1992), and increased risk of meningiomas (which are

mostly benign brain tumors) with early exposures to dental x-rays (BEIR 1990).

In Concord as a whole, female brain cancer was statistically significantly elevated for the period 1982-1992 (13 obs vs 5.4 exp). A review of these female brain cancer cases revealed a variety of different histologic types (e.g., different tissues) and locations of primaries (e.g., different regions) in the brain. In addition, the histologic types found in the different age categories were not unusual for that age group (e.g., medulloblastoma in young individuals). In census tract 3612, female brain cancer was also statistically significantly elevated for the 1982-1992 period (5 obs vs 1.3 exp). These five women had brain cancers of different histologic types and different regions of the brain. Thus, any single factor (e.g., an environmental exposure, a lifestyle factor) is unlikely to explain the significant elevation in either Concord as a whole or in census tract 3612.

Leukemia

Leukemia is a group of malignancies of the white blood cells, classified as separate histological types. Epidemiologic studies have shown that each histologic type of leukemia is an individual disease with specific characteristics, patterns of survival, and etiologic factors. There are known and suspected risk factors for each type of leukemia, however, these account for only a small number of cases (Linet 1985).

Leukemias are classified as either acute or chronic. Ninety percent of children with acute leukemias have acute lymphocytic leukemia (ALL), with a peak incidence of about 4 years old. ALL is the most frequently occurring childhood cancer and accounts for 45 percent of childhood cancers in the U.S. (Page and Asire 1985, Schottenfeld and Fraumeni 1982). Eighty percent of adults with acute leukemias have acute myelocytic leukemia (AML), with the greatest incidence over 65 years of age. ALL also occurs in adults beginning at about the age of 40 and increasing in age thereafter.

Chronic lymphocytic leukemia (CLL) is almost nonexistent before age 30, occurs at an average age of about 65 years, and occurs more often in men than in women. Chronic myelocytic leukemia (CML) accounts for about 20-30 percent of all leukemias, and its incidence peaks in the mid-40s.

The known risk factors for ALL are ionizing radiation and benzene exposure (Linet 1985, Schottenfeld and Fraumeni 1982). Long-term followup of survivors of the atomic bomb explosion at Hiroshima and Nagasaki showed leukemogenic effects (ACS 1996). As a group, the Hiroshima survivors showed increased risk for acute leukemia and for CML (ACS 1996).

A recent study evaluated smoking as a cause of leukemia and found an association with acute myeloid leukemia and adult-age acute lymphocytic leukemia. However, epidemiologic evidence is preliminary and a causal relationship has not been established (Sandler et al. 1992, Siegel 1993).

The only known risk factor for CLL is benzene exposure. Ionizing radiation does not appear to be associated with this disease. Some suspected risk factors for CLL are genetic, immunological, viral, occupational, and environmental (Linet 1985).

Known risk factors for AML are similar to those for ALL: exposure to ionizing radiation and benzene (Schottenfeld and Fraumeni 1982). For example, benzene-exposed shoe, leather, rubber, and chemical

manufacturing workers have shown increased leukemia (primarily AML) (NCI 1996). Suspected risk factors for AML include occupational and environmental exposures and certain drug therapies. Studies suggest that viral and genetic factors play a less important role in the development of AML than in ALL or CLL. Suspected chemical exposures which may suggest an environmental etiology include petroleum products and organic solvents (Linet 1985, Schottenfeld and Fraumeni 1982).

For the five male cases that occurred in census tract 3613 in Concord during 1982-1986 (a statistically significant elevation for this time period), four different types of leukemia (including both acute and chronic types) were observed in these individuals. The fact that these leukemias were different suggest that a common etiology is not likely to explain the observed significant elevation.

Prostate Cancer

Prostate cancer is one of the most common cancers among American men (NCI 1996). It is primarily a disease of the elderly, with a median age of 72 years Studies that show migrant populations tending to prostate cancer risk patterns similar to their host country strongly suggest that environmental factors contribute to differences in incidence patterns among countries (NCI 1996). Among those factors under study are consumption of greater animal fat in the diet and greater dietary intake of vitamin A (NCI 1996). A family history of prostate cancer has also been associated with the disease (Schottenfeld and Fraumeni 1996). Other risk factors that appear weak at best include a history of benign prostate disease, exposure to sexually transmitted agents, or increased risk associated with some occupations, such as farming (NCI 1996). Hormones may also play an important role, and diet or other factors may influence hormonal factors that may play a role in prostate cancer (NCI 1996, ACS 1996).

Estimate of Opportunities for Exposure to Uranium

As an additional evaluation of potential opportunities for offsite exposures to depleted or natural uranium in soil, we also reviewed the available data reported by MDPH/RCP, NRC, and CREW for radioactive contaminants in offsite soil samples. If depleted uranium from the site reached offsite locations, the probable exposure route would be via inhalation of particulates emitted from the facility and from resuspended soil or ingestion of soil that would have been contaminated by particulates that settled from the atmosphere. Skin contact with uranium particulates would not lead to appreciable absorption through intact skin due to the insolubility of the uranium compounds. In animal studies, direct application of insoluble uranium compounds has not shown dermal effects (ATSDR 1990).

MDPH/BEHA asked ATSDR to conduct this further evaluation of potential exposures to uranium in soil reported from NMI-related investigations. ATSDR assumed that a residence could be located where the maximum concentration of U-238 (about 20 pCi/g) in offsite soils was reported. Opportunities for exposure via inhalation of uranium particulates (using data on particulate air quality patterns for the area [U.S. EPA 1995]) and ingestion of soil and indoor housedust were evaluated, assuming that a 2-3 year old pica child (i.e., a child with a craving for unnatural food, e.g., soil) could be living in the residence. The total effective dose to this individual would be less than one millirem per year (mrem/year) or less than 0.01 millisievert per year. (A whole body internal exposure is referred to as an "effective dose," which is the sum of the weighted equivalent doses in all tissues and organs of the body [ICRP 1990]). The International Commission on Radiological Protection (ICRP) recommends to limit the total dose to

a member of the general public to 100 mrem/year (1 mSv/yr) above natural background (ICRP 1978), which is more than one hundred times the estimated opportunity for exposure to uranium in soil. It should be stressed that this evaluation was conducted to determine the likelihood of health effects based on the environmental data reviewed and should in no way be confused with appropriate regulatory cleanup levels.

Limitations

This investigation is descriptive in nature and can only provide a comparison of the incidence of various cancer types in Concord with the incidence of these cancer types in the state. Descriptive assessments have certain inherent limitations. Only routinely collected data are analyzed, and information about personal risk factors (e.g., occupation, diet, smoking), which may influence cancer incidence, is often limited and is not of an historical nature. In many instances, the number of cancer cases was small (sometimes too small to evaluate statistically) and particularly difficult to interpret. It is beyond the scope of this investigation to determine any causal relationship or synergistic roles that the risk factors discussed in this report may have played in the development of the cancer types investigated in Concord. Finally, the reader may want to compare one census tract's SIR with another, or compare an SIR for a cancer type in Concord with an SIR for that cancer type in another town. Such a comparison, however, is not appropriate or meaningful because each census tract is age-adjusted to a different standard (i.e., the state cancer rate and the specific age distribution of the tract's population). The SIR values are imprecise estimates of cancer incidence that serve as indicators of incidence after adjusting for the age distribution of a population. Only small differences in a population's age distribution are sufficient to affect some change in SIR values. Therefore, the comparison of SIRs in tracts with some difference in their age distribution for the purpose of stating which tract has a higher SIR would result in misleading information and possibly incorrect conclusions.

V. CONCLUSIONS

The available data do not show a common pattern that would suggest that any single risk factor is likely to be responsible for the pattern of cancer incidence in Concord. This is not to say that it is not possible that environmental factors may have played a role in the incidence of these cancers but rather that the information reviewed to date does not point to any single factor that may be responsible.

Those cancers (bone and lung) that appear to be most likely associated with the type of radiation emitted by depleted uranium or natural uranium (i.e., primarily alpha particles) occurred about as or less often than expected in Concord and its census tracts. One case of bone cancer (female) occurred townwide during 1982-1992.

Concord or its census tracts did experience some significant elevations in several cancer types during at least one of the time periods reviewed here. Thyroid cancer was most consistently elevated across time periods. Mapping of the cases of each cancer type did not reveal unusual geographic concentrations within census tracts, with the exception of male multiple myeloma in census tract 3611.

A review of the radiological data gathered to date as a result of NMI-related investigations did not seem to suggest that opportunities for exposure to uranium in offsite soils would have likely resulted in

increased cancer incidence or noncancer effects if the soil data collected thus far are representative of soil concentrations throughout the area.

The variety of cancer types and areas of Concord with significant elevations and the general lack of geographic concentrations of any cancer types (examined and mapped on a scale less than the census tract) do not suggest a single risk factor can explain the observed cancer incidence.

VI. RECOMMENDATIONS

1. MDPH/BEHA will further investigate the multiple myeloma cases in census tract 3611 and thyroid cancer cases in Concord by reviewing the residential histories of these cases and, if necessary, medical information on the cases.

2. The MDPH/BEHA will prepare a separate response addressing a citizen concern received after initiation of this investigation regarding testicular cancer and melanoma in Concord.

3. The MDPH/BEHA will continue to monitor cancer incidence rates in Concord through the Massachusetts Cancer Registry.

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TABLE 1. TOWNWIDE CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1986

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	38	55.5	68*	48-94	22	36.8	60*	37-91	16	18.8	85	49-138
bone	1	0.7	NC	NC	0	0.4	--	--	1	0.3	NC	NC
thyroid	6	2.9	208	76-453	2	1.1	NC	NC	4	1.8	NC	NC
multiple myeloma	3	3.4	NC	NC	1	1.7	NC	NC	2	1.7	NC	NC
brain	6	5.2	115	42-250	2	3.0	NC	NC	4	2.3	NC	NC
breast	83	56.6	147*	117-182	1	0.4	NC	NC	82	56.2	146*	116-181
leukemia	10	7.7	130	62-239	8	4.2	190	82-375	2	3.3	NC	NC
prostate	25	32.4	77	50-114	25	32.4	77	50-114	--	--	--	--
colorectal	59	62.1	95	72-123	29	30.9	94	63-135	30	31.2	96	65-137

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 2. TOWNWIDE CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1987-1992

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	53	71.5	74*	56-97	27	43.7	62*	41-90	26	27.8	93	61-137
bone	0	0.9	--	--	0	0.5	--	--	0	0.4	--	--
thyroid	10	4.0	247*	118-455	4	1.3	NC	NC	6	2.8	216	79-469
multiple myeloma	9	4.2	216	98-409	9	2.2	412*	188-782	0	2.0	--	--
brain	13	7.2	180	96-307	4	4.1	NC	NC	9	3.2	286*	130-542
breast	76	79.0	96	76-120	1	0.5	NC	NC	75	78.5	96	75-120
leukemia	12	8.4	143	74-250	6	4.9	123	45-268	6	3.5	171	62-371
prostate	73	56.7	129*	101-162	73	56.7	129*	101-162	--	--	--	--
colorectal	70	70.0	100	78-126	34	35.8	95	66-133	36	34.2	105	74-146

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 3. TOWNWIDE CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1992

	Total				Male				Female			
Cancer Type	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	91	127.3	72*	58-88	49	80.6	61*	45-80	42	46.7	90	65-122
bone	1	1.6	NC	NC	0	0.8	--	--	1	0.7	NC	NC
thyroid	16	6.9	230*	132-374	6	2.3	256	94-557	10	4.6	217*	104-399
multiple myeloma	12	7.6	158	81-276	10	3.9	258*	124-475	2	3.7	NC	NC
brain	19	12.4	154	93-240	6	7.0	86	31-187	13	5.4	242*	129-414
breast	159	135.7	117	100-137	2	0.9	NC	NC	157	134.8	116	99-136
leukemia	22	15.9	138	87-209	14	9.1	154	84-259	8	6.8	117	50-231
prostate	98	89.7	109	89-133	98	89.7	109	89-133	--	--	--	--
colorectal	129	132.3	98	81-116	63	66.7	94	73-121	66	65.5	101	78-128

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance

Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 4. CENSUS TRACT 3611 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1986

	Total				Male				Female			
Cancer Type	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	15	21.5	70	39-115	10	14.1	71	34-131	5	7.4	67	22-157
bone	0	0.3	--	--	0	0.1	--	--	0	0.1	--	--
thyroid	2	1.1	NC	NC	0	0.4	--	--	2	0.7	NC	NC
multiple myeloma	1	1.4	NC	NC	1	0.7	NC	NC	0	0.8	--	--
brain	3	1.9	NC	NC	1	1.0	NC	NC	2	0.8	NC	NC
breast	30	23.0	130	88-186	1	0.1	NC	NC	29	22.9	127	85-182
leukemia	4	3.1	NC	NC	2	1.6	NC	NC	2	1.4	NC	NC
prostate	13	12.6	103	55-176	13	12.6	103	55-176	--	--	--	--
colorectal	19	26.5	72	43-112	8	12.2	65	28-129	11	14.3	77	38-138

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 5. CENSUS TRACT 3611 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1987-1992

	Total				Male				Female			
Cancer Type	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	17	27.8	61*	36-98	7	16.7	42*	17-86	10	11.1	90	43-166
bone	0	0.3	--	--	0	0.2	--	--	0	0.1	--	--
thyroid	5	1.5	334*	108-779	1	0.5	NC	NC	4	1.0	NC	NC
multiple myeloma	4	1.7	NC	NC	4	0.9	NC	NC	0	0.9	--	--
brain	2	2.5	NC	NC	0	1.5	--	--	2	1.1	NC	NC
breast	37	31.7	117	82-161	0	0.2	--	--	37	31.4	118	83-162
leukemia	5	3.4	148	48-346	3	1.9	NC	NC	2	1.5	NC	NC
prostate	31	22.7	137	93-194	31	22.7	137	93-194	--	--	--	--
colorectal	25	29.9	84	54-124	10	14.2	70	34-130	15	15.7	96	54-158

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance

Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 6. CENSUS TRACT 3611 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1992

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	32	49.4	65*	44-91	17	30.8	55*	32-88	15	18.6	81	45-133
bone	0	0.6	--	--	0	0.3	--	--	0	0.3	--	--
thyroid	7	2.6	269*	108-555	1	0.9	NC	NC	6	1.7	356*	130-776
multiple myeloma	5	3.1	158	51-369	5	1.5	327*	106-764	0	1.6	--	--
brain	5	4.7	106	34-247	1	2.7	NC	NC	4	2.0	NC	NC
breast	67	54.7	122	95-155	1	0.3	NC	NC	66	54.4	121	94-154
leukemia	9	6.4	140	64-266	5	3.5	142	46-331	4	2.9	NC	NC
prostate	44	36.1	122	88-164	44	36.1	122	88-164	--	--	--	--
colorectal	44	56.4	78	57-105	18	26.4	68	40-108	26	30.0	87	57-127

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 7. CENSUS TRACT 3612 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1986

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	10	13.5	74	36-136	4	9.1	NC	NC	6	4.4	137	50-299
bone	1	0.2	NC	NC	0	0.1	--	--	1	0.1	NC	NC
thyroid	0	0.8	--	--	0	0.3	--	--	0	0.5	--	--
multiple myeloma	1	0.7	NC	NC	0	0.4	--	--	1	0.3	NC	NC
brain	2	1.3	NC	NC	1	0.8	NC	NC	1	0.6	NC	NC
breast	24	13.0	185*	118-275	0	0.1	--	--	24	12.9	186*	119-277
leukemia	1	2.0	NC	NC	1	1.2	NC	NC	0	0.8	--	--
prostate	4	7.1	NC	NC	4	7.1	NC	NC	--	--	--	--
colorectal	13	13.2	99	53-169	8	7.4	109	47-214	5	5.8	86	28-201

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 8. CENSUS TRACT 3612 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1987-1992

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	16	17.2	93	53-151	11	10.8	102	51-182	5	6.4	79	25-183
bone	0	0.2	--	--	0	0.1	--	--	0	0.1	--	--
thyroid	4	1.1	NC	NC	2	0.3	NC	NC	2	0.8	NC	NC
multiple myeloma	1	0.9	NC	NC	1	0.5	NC	NC	0	0.4	--	--
brain	5	1.9	270	87-630	1	1.1	NC	NC	4	0.8	NC	NC
breast	13	18.4	71	38-121	1	0.1	NC	NC	12	18.3	66	34-114
leukemia	5	2.0	250	80-583	1	1.2	NC	NC	4	0.8	NC	NC
prostate	17	13.1	129	75-207	17	13.1	129	75-207	--	--	--	--
colorectal	19	14.9	128	77-199	11	8.5	129	64-231	8	6.4	126	54-248

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 9. CENSUS TRACT 3612 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1992

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	26	30.7	85	55-124	15	19.9	75	42-124	11	10.7	102	51-183
bone	1	0.4	NC	NC	0	0.2	--	--	1	0.2	NC	NC
thyroid	4	1.9	NC	NC	2	0.6	NC	NC	2	1.3	NC	NC
multiple myeloma	2	1.7	NC	NC	1	0.9	NC	NC	1	0.7	NC	NC
brain	7	3.2	219	88-450	2	1.9	NC	NC	5	1.3	371*	119-865
breast	37	31.4	118	83-162	1	0.2	NC	NC	36	31.2	115	81-160
leukemia	6	3.8	159	58-346	2	2.3	NC	NC	4	1.5	NC	NC
prostate	21	20.6	102	63-156	21	20.6	102	63-156	--	--	--	--
colorectal	32	28.1	114	78-161	19	15.9	120	72-187	13	12.2	107	57-182

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 10. CENSUS TRACT 3613 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1986

	Total				Male				Female			
Cancer Type	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	13	20.6	63	34-108	8	13.6	59	25-116	5	7.0	72	23-167
bone	0	0.2	--	--	0	0.1	--	--	0	0.1	--	--
thyroid	4	1.0	NC	NC	2	0.4	NC	NC	2	0.7	NC	NC
multiple myeloma	1	1.2	NC	NC	0	0.6	--	--	1	0.6	NC	NC
brain	1	1.9	NC	NC	0	1.1	--	--	1	0.8	NC	NC
breast	29	20.7	140	94-202	0	0.1	--	--	29	20.5	141	95-203
leukemia	5	2.7	186	60-433	5	1.5	333*	107-777	0	1.2	--	--
prostate	8	11.4	70	30-138	8	11.4	70	30-138	--	--	--	--
colorectal	27	22.5	120	79-175	13	11.3	115	61-197	14	11.2	125	68-210

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 11. CENSUS TRACT 3613 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1987-1992

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	18	26.5	68	40-107	8	16.1	50*	21-98	10	10.4	96	46-177
bone	0	0.3	--	--	0	0.2	--	--	0	0.1	--	--
thyroid	1	1.5	NC	NC	1	0.4	NC	NC	0	1.0	--	--
multiple myeloma	3	1.5	NC	NC	3	0.8	NC	NC	0	0.7	--	--
brain	6	2.6	228	83-497	3	1.5	NC	NC	3	1.2	NC	NC
breast	26	29.0	90	58-131	0	0.2	--	--	26	28.8	90	59-132
leukemia	2	3.0	NC	NC	2	1.7	NC	NC	0	1.3	--	--
prostate	24	20.9	115	74-171	24	20.9	115	74-171	--	--	--	--
colorectal	26	25.3	103	67-151	13	13.1	100	53-170	13	12.3	106	56-181

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 12. CENSUS TRACT 3613 CANCER INCIDENCE—CONCORD, MASSACHUSETTS 1982-1992

Cancer Type	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
lung	31	47.2	66*	45-93	16	29.8	54*	31-87	15	17.5	86	48-142
bone	0	0.6	--	--	0	0.3	--	--	0	0.3	--	--
thyroid	5	2.5	201	65-469	3	0.8	NC	NC	2	1.7	NC	NC
multiple myeloma	4	2.8	NC	NC	3	1.4	NC	NC	1	1.4	NC	NC
brain	7	4.5	155	62-319	3	2.5	NC	NC	4	2.0	NC	NC
breast	55	49.7	111	83-144	0	0.3	--	--	55	49.4	111	84-145
leukemia	7	5.7	123	49-253	7	3.3	215	86-443	0	2.5	--	--
prostate	32	32.9	97	66-137	32	32.9	97	66-137	--	--	--	--
colorectal	53	47.8	111	83-145	26	24.4	107	70-156	27	23.5	115	76-167

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth
Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance
Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 13. THYROID CANCER INCIDENCE—ACTON, MAYNARD, AND SUDBURY, MASSACHUSETTS 1982-1992

	Total				Male				Female			
	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI	Obs	Exp	SIR	CI
Acton												
1982-1986	1	2.8	NC	NC	0	1.0	--	--	1	1.8	NC	NC
1987-1992	8	4.0	198	85-391	5	1.1	453*	146-1058	3	2.9	NC	NC
1982-1992	9	6.9	131	60-249	5	2.1	243	78-568	4	4.8	NC	NC
Maynard												
1982-1986	1	1.7	NC	NC	0	0.6	--	--	1	1.1	NC	NC
1987-1992	1	2.4	NC	NC	0	0.7	--	--	1	1.7	NC	NC
1982-1992	2	4.1	NC	NC	0	1.3	--	--	2	2.8	NC	NC
Sudbury												
1982-1986	2	2.2	NC	NC	1	0.8	NC	NC	1	1.4	NC	NC
1987-1992	4	3.2	NC	NC	0	0.9	--	--	4	2.3	NC	NC
1982-1992	6	5.5	110	40-239	1	1.7	NC	NC	5	3.7	134	43-313

SIR standardized incidence ratio; calculated based on the exact number of expected cases; expected cases presented here are rounded to the nearest tenth

Obs observed number of cases
Exp expected number of cases
CI 95-percent confidence interval for SIR
NC not calculated when observed number of cases is less than 5
* indicates statistical significance

Data source is Massachusetts Cancer Registry, Bureau of Health Statistics, Research, and Evaluation, Massachusetts Department of Public Health

TABLE 14. SIGNIFICANT ELEVATIONS
CANCER INCIDENCE IN CONCORD, MASSACHUSETTS

	1982-1986	1987-1992	1982-1992
Thyroid			
Concord		total	total female
CT 3611		total	female
Multiple myeloma			
Concord		male	male
CT 3611			male
Breast			
Concord	female		
CT 3612	female		
Brain			
Concord		female	female
CT 3612			female
Leukemia			
CT 3613	male		
Prostate			
Concord		male	

FIGURE 1

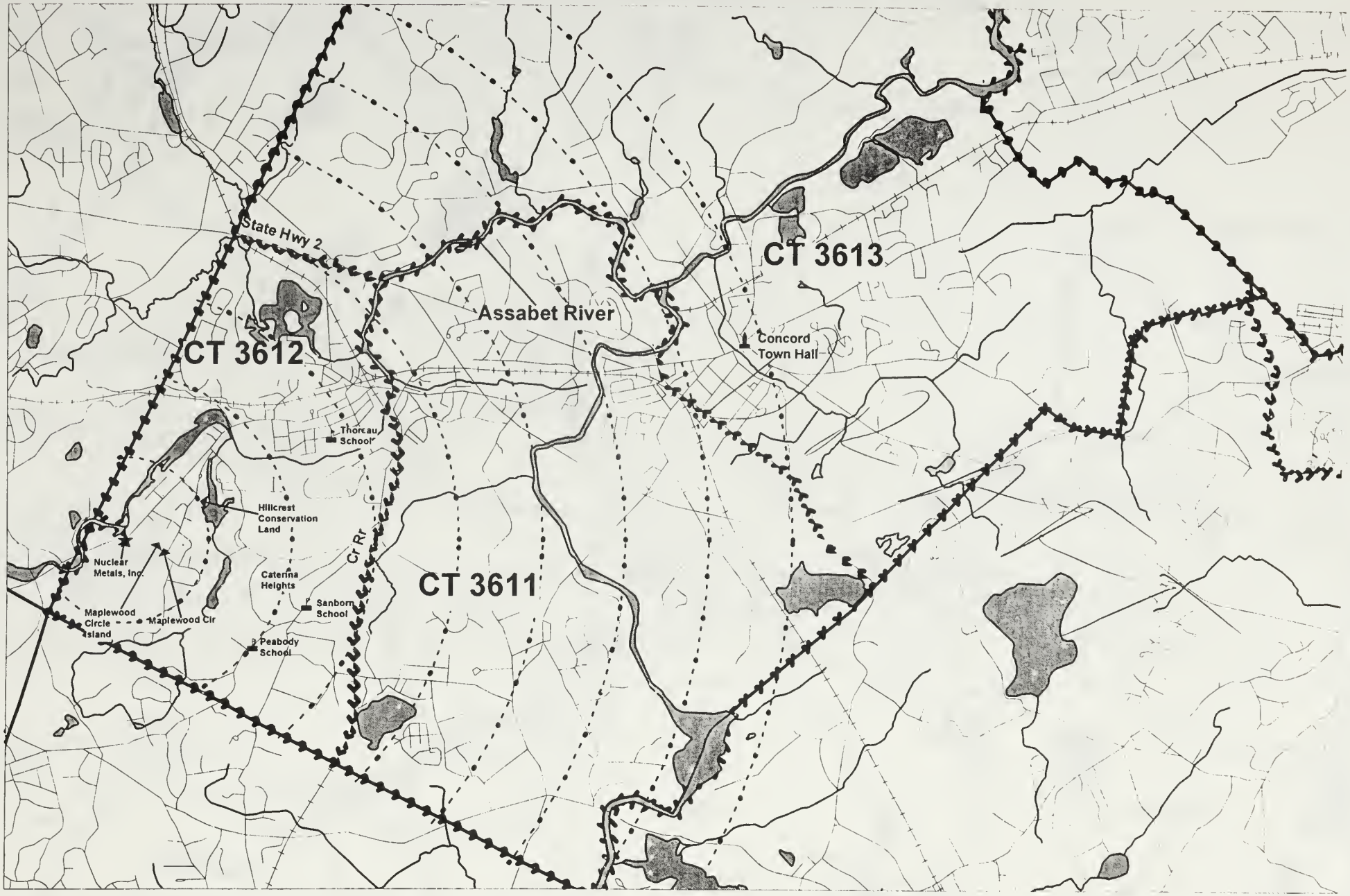
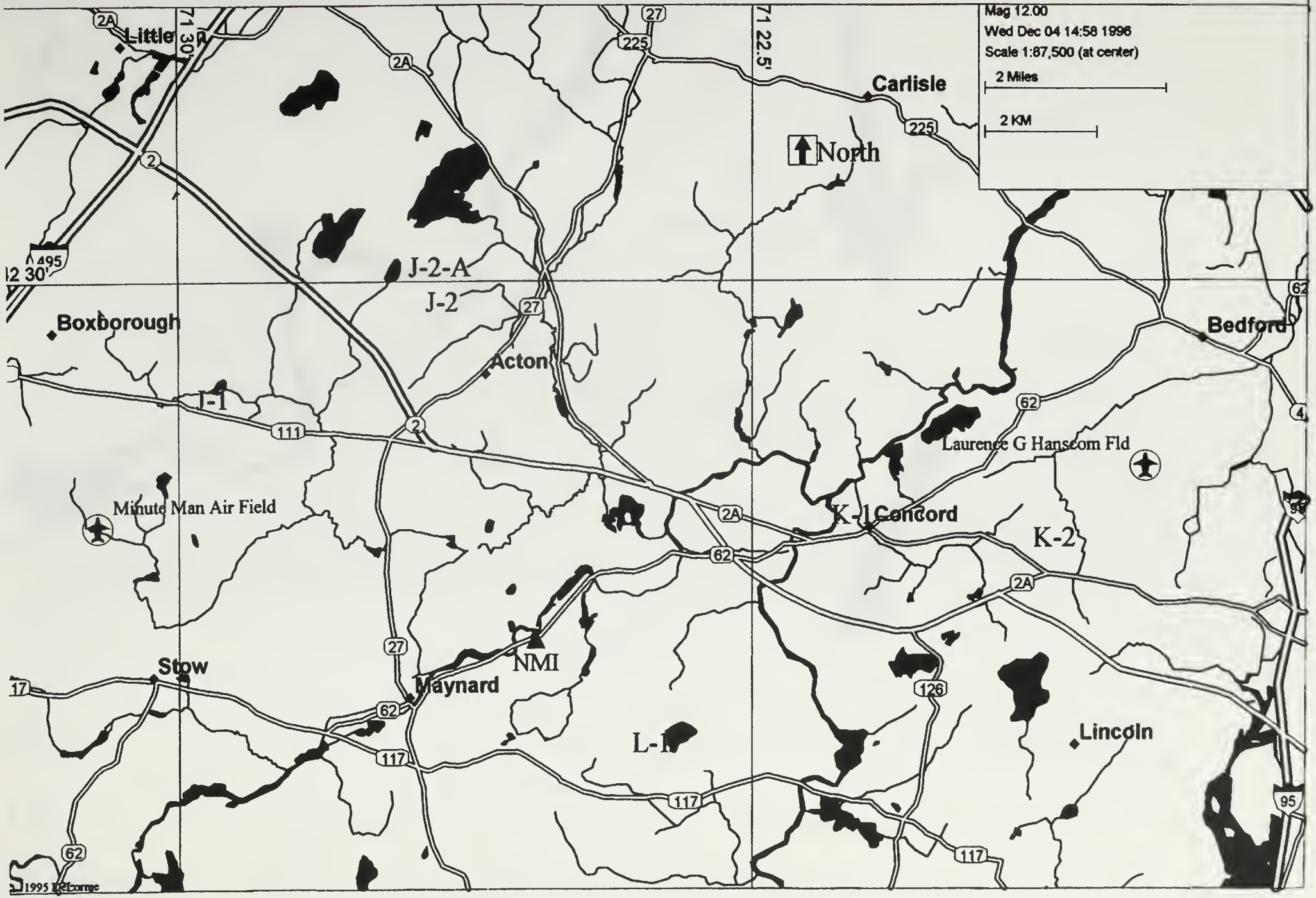
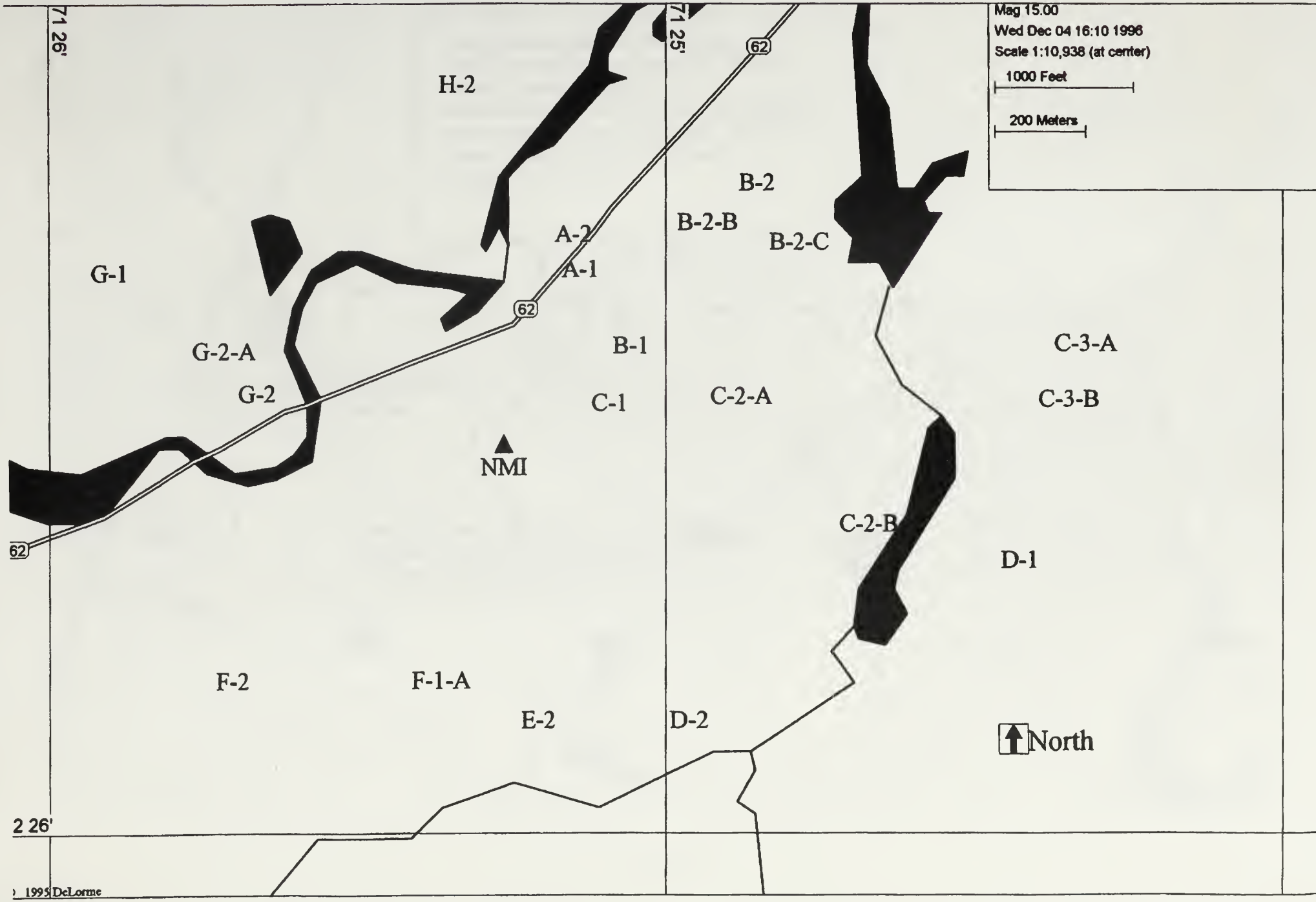

State Hwy 2
Assabet River
CT 3613
CT 3612
CT 3611
Concord
Town Hall
Thoreau
School
Hillcrest
Conservation
Land
Cr Rr
Nuclear
Metals, Inc.
Caterina
Heights
Sanborn
School
Maplewood
Circle
Island
Maplewood Cir
Peabody
School

FIGURE 2

NMI Soil Sampling map 1



NMI Soil Sampling map 2



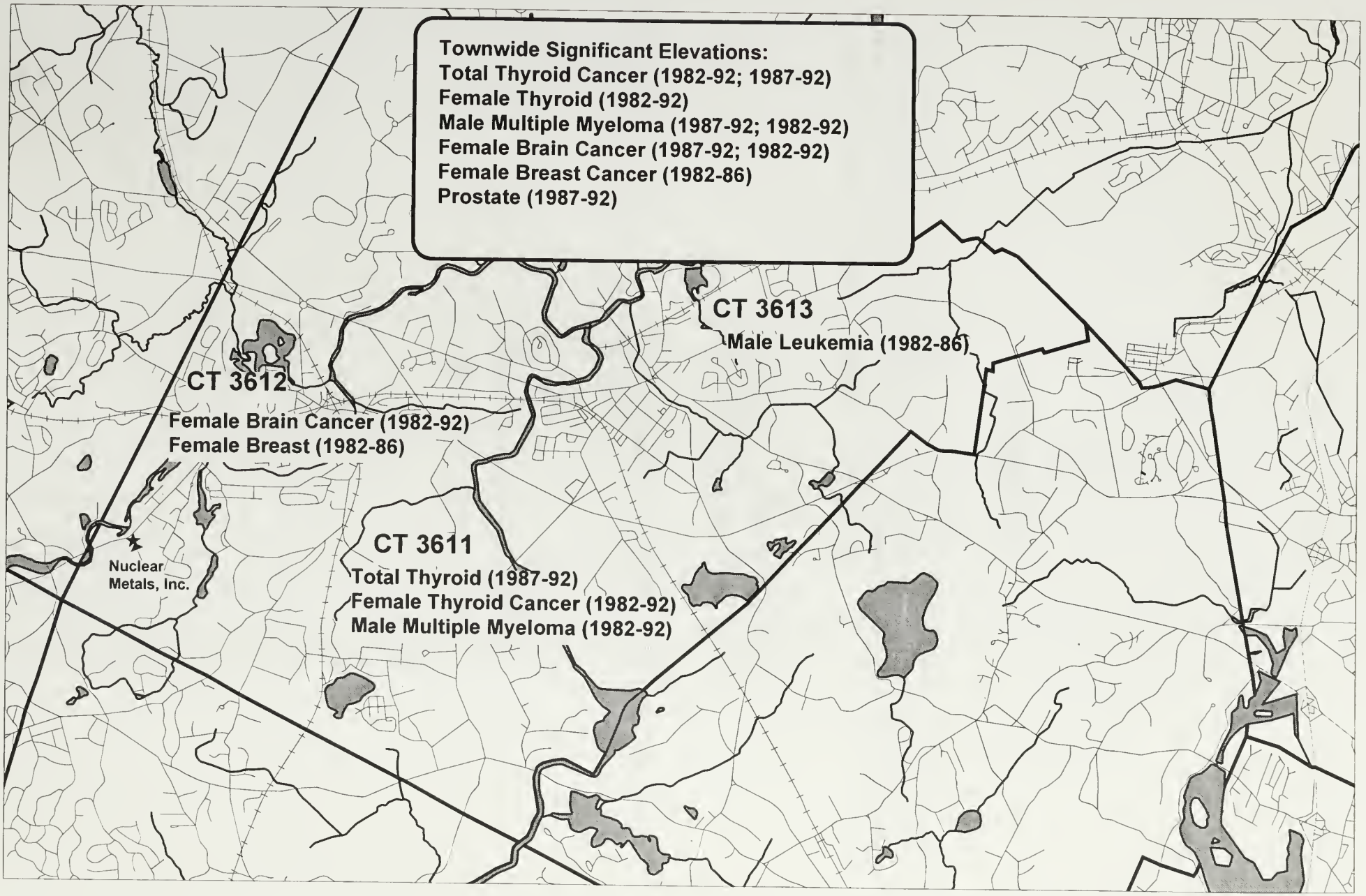

Townwide Significant Elevations:
Total Thyroid Cancer (1982-92; 1987-92)
Female Thyroid (1982-92)
Male Multiple Myeloma (1987-92; 1982-92)
Female Brain Cancer (1987-92; 1982-92)
Female Breast Cancer (1982-86)
Prostate (1987-92)
CT 3613
Male Leukemia (1982-86)
CT 3612
Female Brain Cancer (1982-92)
Female Breast (1982-86)
Nuclear Metals, Inc.
CT 3611
Total Thyroid (1987-92)
Female Thyroid Cancer (1982-92)
Male Multiple Myeloma (1982-92)

FIGURE 4

CERTIFICATION

The Nuclear Metals Health Consultation was prepared by the Massachusetts Department of Public Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures existing at the time the Health Consultation was initiated.

Technical Project Officer, SPS, SSAB, DHAC

The Division of Health Assessment and Consultation, ATSDR, has reviewed this Health Consultation and concurs with its findings.

Chief, SPS, SSAB, DHAC, ATSDR